Glaucoma: the “silent thief of sight”

In this article...
- Glaucoma and how its progression can be prevented
- The role of the nurse

Glaucoma has been called the “silent thief of sight” as it is asymptomatic but causes irreversible vision loss. One of the most common ophthalmic conditions in the world, it is also the leading cause of irreversible blindness (World Health Organization, 2010). In England and Wales, it is the second most common cause for registration of visual impairment (Bunce et al, 2010). Glaucoma is an umbrella term for a large group of disorders characterised by diverse clinical and pathological features. The common characteristics are:
- Optic nerve damage;
- Visual field loss; and
- Irreversible blindness.

It is a silent progressive disease and is one of the leading causes of preventable blindness or significant visual impairment if arrested before significant effects occur. Screening is key to diagnosis, and treatment adherence is critical to prevent vision loss in those who are diagnosed. Nurses have vital roles in both these areas.

Glaucoma accounts for a major proportion of the workload – one million visits a year – in ophthalmology settings in the UK. Around 2% of people aged over 40 years and almost 10% of those aged over 75 have primary (chronic) open-angle glaucoma (POAG); only about half are diagnosed (National Institute for Health and Care Excellence, 2009).

The social and economic burden of reduced vision due to glaucoma and the ophthalmology workload is likely to grow, due to increasing life expectancy (Coleman and Miglior, 2008; Burr et al, 2007). As glaucoma is asymptomatic until vision has been lost, patients may present with significant irreversible changes. Once vision loss becomes apparent, 90% of optic nerve fibres may have become irreparably damaged (NICE, 2009). Treatment slows progression by lowering intraocular pressure (IOP).

What is glaucoma?
Glaucoma is initially classified depending on whether the drainage angle of the eye is open (open-angle) or closed (angle closure or acute). Open-angle glaucoma is further categorised into primary (idiopathic) and secondary (associated with comorbidities such as inflammation and neovascularisation) glaucoma. This article focuses on POAG, which accounts for the majority of glaucoma seen in practice (King et al, 2013).

Pathophysiology
The pathophysiology of POAG is not well understood (King et al, 2013). A raised IOP is common and thought to be due to a resistance to outflow in the trabecular meshwork. Loss of vision is due to retinal cell death; the main site of nerve damage is thought to be the optic nerve head, where all axons of the retinal cells exit the eye.

A number of factors may contribute to cell damage, including mechanical pressure on the nerve cells and poor perfusion and ischaemia of the cells in this area of the retina.

By adhering to treatment patients will have an increased chance of retaining their sight.
The major risk factors for glaucoma are:

**Predisposing factors for glaucoma**

- Some types of secondary open-angle glaucoma progress slowly, over years, although a significant level of deterioration usually may not be noticed until it has reached a minor role peripheral vision plays in most of our eyes’ overlapping visual fields and the temporary field is generally damaged first. Due to damage to the field of vision; the peripheral vision loss may not be noticed until it has reached a minor role.

**Consequences of glaucoma**

- Intraocular pressure
- Aqueous humour (aqueous) is a clear fluid produced by the non-pigmented portion of the ciliary processes through active secretion, ultra filtration and diffusion. It flows into the posterior chamber, through the pupil into the anterior chamber. Although derived from plasma, aqueous humour contains no protein, consisting mainly of water with electrolytes, glucose, amino acids, a high concentration of ascorbic acid and dissolved gases. It provides nourishment for the posterior cornea and lens as well maintaining the shape of the eyeball.

- The level of IOP depends on the amount of aqueous humour secreted and drained effectively; 90% is drained through the trabecular meshwork, the rest flows across the ciliary body, through the suprachoroidal space and into the venous circulation.

- **Consequences of glaucoma**

  **Damage to retinal nerve cells results in damage to the field of vision; the peripheral field is generally damaged first. Due to our eyes’ overlapping visual fields and the minor role peripheral vision plays in most people’s perception of what they see, sight loss may not be noticed until it has reached a significant level. Deterioration usually progresses slowly, over years, although some types of secondary open-angle glaucoma may progress more rapidly.**

**Predisposing factors for glaucoma**

- The major risk factors for glaucoma are:

  - Age: incidence increases with age, most commonly presenting after the age of 65 and rarely before the age of 40 years;
  - Family history: one of the strongest risk factors, risk is stronger in siblings than offspring. A genetic basis for glaucoma has been suggested but it is felt this is only likely to be true in a few cases;
  - Race: the Baltimore Eye Survey (Tielsch et al, 1994) showed a three to four-fold higher prevalence of glaucoma in African-Americans than Caucasians, for every age group. Glaucoma tends to progress more rapidly in this group as well; and
  - Raised IOP.

**Diagnosis and detection**

- It is estimated that more than half the people with glaucoma in the developed world and 90% in the developing world have not been diagnosed (Wittenborn and Rein, 2011; Burr et al, 2007). Detection is often opportunistically, through community optometric services. Unequal access to these facilities means those with low incomes are less likely to be identified and treated. However, access to community optometry does not guarantee detection; in the UK, many of those referred by optometrists for investigation do not have the condition (King et al, 2013), while around 50% of those newly diagnosed in one study (Vahtoranta-Lehtonen et al, 2007) had been seen by an optometrist or ophthalmologist, without glaucoma being identified.

  - Patients are often referred with a raised IOP but this is not diagnostic of glaucoma. Evaluation of these patients includes:

    - Measurement of IOP using a Goldmann applanation tonometer mounted on a slit lamp;
    - Measurement of the central corneal thickness (a thicker cornea may not be as indentable as a cornea of normal thickness, so pressure inside the eye may be as high as it appears. Conversely, a thinner cornea may give an inaccurate low pressure reading); and
    - Visualisation of the drainage angle using a special contact lens known as a gonioscopy lens;

- **Intraocular pressure**

  Aqueous humour (aqueous) is a clear fluid produced by the non-pigmented portion of the ciliary processes through active secretion, ultra filtration and diffusion. It flows into the posterior chamber, through the pupil into the anterior chamber. Although derived from plasma, aqueous humour contains no protein, consisting mainly of water with electrolytes, glucose, amino acids, a high concentration of ascorbic acid and dissolved gases. It provides nourishment for the posterior cornea and lens as well maintaining the shape of the eyeball.

- **Systemic side-effects**

  - (Rarely) hypotension, bradycardia, brow ache

- **Ocular side-effects**

  - Change in eye colour, brown pigmentation of skin, thickening and lengthening of eyelashes

**Drug type**

<table>
<thead>
<tr>
<th>Drug type</th>
<th>Prostaglandin analogues</th>
<th>Beta blockers</th>
<th>Carbonic anhydrase inhibitors</th>
<th>Sympathomimetics</th>
<th>Miotics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Action</strong></td>
<td>Increase aqueous outflow</td>
<td>Reduce aqueous secretion</td>
<td>Reduce aqueous secretion</td>
<td>Reduce aqueous secretion and increase outflow</td>
<td>Increase aqueous outflow</td>
</tr>
<tr>
<td><strong>Ocular side-effects</strong></td>
<td>Change in eye colour, brown pigmentation of skin, thickening and lengthening of eyelashes</td>
<td>Irritation, erythema, dry eyes, blepharoconjunctivitis and allergy (anaphylactic reaction possible)</td>
<td>Localised discomfort, lacrimation, topical allergy</td>
<td>Mydriasis (dilated pupils), dry eye, severe smarting and redness of the eye</td>
<td>Miosis (contraction of the pupil) causing blurred vision. Brow ache, pupillary block, localised discomfort</td>
</tr>
<tr>
<td><strong>Systemic side-effects</strong></td>
<td>(Rarely) hypotension, bradycardia, brow ache</td>
<td>Bronchospasam, bradycardia, exacerbation of heart failure, nightmares</td>
<td>Taste disturbance, nausea/vomiting, headache, dizziness, fatigue, paraesthesia</td>
<td>Lethargy, hypotension</td>
<td>Sweating, bradycardia, gastrointestinal disturbance</td>
</tr>
</tbody>
</table>

**Monitoring**

- POAG is a lifelong condition with a varied course. Monitoring is required to ensure control is stable, note changes and implement treatment changes. Complete, accurate notes must be available at every clinic visit to ensure transfer of information between the community and hospital ophthalmic services, and continuity of care.

**Treatment**

- Treatment is aimed at achieving stability, defined as no evidence of progression or progression at a rate at which visual
Impairment does not affect quality of life (including the ability to drive). If glaucoma is not progressing or is doing so very slowly, no treatment may be needed; in other patients, aggressive or rapidly progressing disease can make treatment difficult. Treatment focuses on reducing IOP as this is the modifiable risk to retinal damage.

Once a patient is diagnosed, a target IOP is established; this is the level the clinician believes is low enough to prevent disease progressing to a level that would impair quality of life. Younger patients are likely to have to live with glaucoma for longer and so are given a lower target IOP than older patients. Some, particularly older patients with very slow-progressing disease, may be monitored without treatment as the disease is unlikely to cause disability in their lifetime. Progression at any age will necessitate changes in treatment.

Lack of progression in patients who are very old may prompt talk of discontinuing treatment. This is not related to cost but is a pragmatic strategy based on the likelihood of progression occurring in the patient’s lifespan. Benefits of reducing or stopping treatment may include removing the burden of regular medication.

Drug therapy is the usual first treatment of choice; patients are started on single eye-drop therapy, with extra drops added if the IOP does not reach the target. If a patient is on the maximum tolerated treatment, laser or surgical options may be considered. Treatment pathways are informed by NICE guidance (2009).

Drug treatment

The first-line treatment is usually prosta
glandin analogue drops, which reduce IOP by increasing outflow of aqueous humour via the uveoscleral route (which usually only accounts for 10% of drainage). It may affect pigmentation of the iris and periorbital skin, and lash growth. These drops have the fewest side-effects of the drugs used to treat glaucoma.

Second-line treatment is often the addition of a beta blocker to reduce aqueous humour production. Prostaglandin analogues and beta blockers may be given together to reduce the number of drops the patient must instil. Combination therapy also minimises the amount of preservative entering the eye, thus reducing the risk of intolerance or allergic reactions. Preservative-free drops are also available.

All drugs have contraindications and side-effects, and regimens may change depending on the patient’s systemic and ocular responses (Patient.co.uk, 2014). Table 1 outlines ophthalmic drugs to treat POAG.

Patients at risk of sight loss despite treatment may have surgery to form an artificial drainage channel (trabecuoplasty). As surgical wounds tend to want to heal, patients are given cytotoxic drugs such as mitomycin C, which aims to stop the surgical drainage channel from healing.

Patient information and adherence to treatment

Most patients with glaucoma are asymptomatic. They are referred for investigations at a routine appointment, go to the hospital eye service, have several tests and then remain in the system, still with no symptoms, for life. They are given powerful medicines that may sting, make their eyes sore and blur their vision for a while, and are told it is important they continue to take them. They may do so but if nothing seems to happen – it’s no surprise that adherence to treatment is poor.

If the IOP is not controlled, more drugs are added; if these do not work, surgery may be necessary. However, it is difficult to know whether the failure to control IOP is due to ineffective drugs or non-adherence.

Many factors contribute to non-adherence to treatment. Waterman et al (2013) suggest that simplifying drop regimens, teaching drop instillation and providing information and ongoing support might help improve adherence. Understanding patients’ lifestyles may lead, for example, to prescriptions of single-dose, sustained-release preparations that facilitate adherence. Patients’ recognition of the seriousness of their diagnosis is crucial. Information and support is widely available, but they need to be directed to it. Organisations such as the International Glaucoma Association provide excellent and accessible patient information. Nurses must be aware of all medications used by their patients. Eye drops are often overlooked, but are powerful and can interact with systemic preparations. Beta blockers can cause breathlessness and hypotension, for example, and changes in medication should be monitored. Eye drops are never prescribed for non-therapeutic reasons so if a patient has been prescribed them, they should be given as prescribed.

Conclusion

Patients with long-term conditions such as glaucoma should work in partnership with their care team, be given and take appropriate responsibility, and feel that things are being “done with”, not “done to” them. Key to stopping irreversible and preventable sight loss is detecting those who are at risk of glaucoma and encouraging them to be screened. Providing optometry services for all residents of residential and nursing homes is also important.

Recognising that eye drops are powerful drugs that can reduce the burden of avoidable blindness is also crucial. The therapies available for glaucoma can prevent or slow vision loss, but once vision has been lost it cannot be restored.

References

Patient.co.uk (2014) Primary Open-angle Glaucoma. tinyurl.com/PatientGlaucoma

For more on this topic go online...

- Patient support to reduce risk of diabetic retinopathy
- Bit.ly/NTRetinopathyRisks

Key messages

- People aged over 40 should be encouraged to have regular eye tests
- People with a low income may be able to get help for optometry services. Eye tests in Scotland are free for all, and in England they are free for people over retirement age or those aged 40 or over who are first-degree relatives of someone with glaucoma
- 10% of people aged over 75 have glaucoma, regardless of whether it has been diagnosed.
- If your patient population does not reflect this, highlight screening opportunities
- Teaching drop instillation and providing support may improve treatment adherence
- Once vision has been lost, it cannot be restored

Box 1: Key Messages

- People aged over 40 should be encouraged to have regular eye tests
- People with a low income may be able to get help for optometry services. Eye tests in Scotland are free for all, and in England they are free for people over retirement age or those aged 40 or over who are first-degree relatives of someone with glaucoma
- 10% of people aged over 75 have glaucoma, regardless of whether it has been diagnosed.
- If your patient population does not reflect this, highlight screening opportunities
- Teaching drop instillation and providing support may improve treatment adherence
- Once vision has been lost, it cannot be restored

Drug therapy is the usual first treatment of choice; patients are started on single eye-drop therapy, with extra drops added if the IOP does not reach the target. If a patient is on the maximum tolerable treatment, laser or surgical options may be considered. Treatment pathways are informed by NICE guidance (2009).

Drug treatment

The first-line treatment is usually prostaglandin analogue drops, which reduce IOP by increasing outflow of aqueous humour via the uveoscleral route (which usually only accounts for 10% of drainage). It may affect pigmentation of the iris and periorbital skin, and lash growth. These drops have the fewest side-effects of the drugs used to treat glaucoma.

Second-line treatment is often the addition of a beta blocker to reduce aqueous humour production. Prostaglandin analogues and beta blockers may be given together to reduce the number of drops the patient must instil. Combination therapy also minimises the amount of preservative entering the eye, thus reducing the risk of intolerance or allergic reactions. Preservative-free drops are also available.

All drugs have contraindications and side-effects, and regimens may change depending on the patient’s systemic and ocular responses (Patient.co.uk, 2014). Table 1 outlines ophthalmic drugs to treat POAG.

Patients at risk of sight loss despite treatment may have surgery to form an artificial drainage channel (trabecuoplasty). As surgical wounds tend to want to heal, patients are given cytotoxic drugs such as mitomycin C, which aims to stop the surgical drainage channel from healing.

Patient information and adherence to treatment

Almost all patients with glaucoma are asymptomatic. They are referred for investigations at a routine appointment, go to the hospital eye service, have several tests and then remain in the system, still with no symptoms, for life. They are given powerful medicines that may sting, make their eyes sore and blur their vision for a while, and are told it is important they continue to take them. They may do so but if nothing seems to happen – it’s no surprise that adherence to treatment is poor.

If the IOP is not controlled, more drugs are added; if these do not work, surgery may be necessary. However, it is difficult to know whether the failure to control IOP is due to ineffective drugs or non-adherence.

Many factors contribute to non-adherence to treatment. Waterman et al (2013) suggest that simplifying drop regimens, teaching drop instillation and providing information and ongoing support might help improve adherence. Understanding patients’ lifestyles may lead, for example, to prescriptions of single-dose, sustained-release preparations that facilitate adherence. Patients’ recognition of the seriousness of their diagnosis is crucial. Information and support is widely available, but they need to be directed to it. Organisations such as the International Glaucoma Association provide excellent and accessible patient information. Nurses must be aware of all medications used by their patients. Eye drops are often overlooked, but are powerful and can interact with systemic preparations. Beta blockers can cause breathlessness and hypotension, for example, and changes in medication should be monitored. Eye drops are never prescribed for non-therapeutic reasons so if a patient has been prescribed them, they should be given as prescribed.

Conclusion

Patients with long-term conditions such as glaucoma should work in partnership with their care team, be given and take appropriate responsibility, and feel that things are being “done with”, not “done to” them. Key to stopping irreversible and preventable sight loss is detecting those who are at risk of glaucoma and encouraging them to be screened. Providing optometry services for all residents of residential and nursing homes is also important.

Recognising that eye drops are powerful drugs that can reduce the burden of avoidable blindness is also crucial. The therapies available for glaucoma can prevent or slow vision loss, but once vision has been lost it cannot be restored.